Development of Sclerotinia Stem Rot in Potato Fields in South-Central Washington

Z. K. Atallah and D. A. Johnson, Department of Plant Pathology, Washington State University, Pullman 99164-6430

ABSTRACT

Atallah, Z. K., and Johnson, D. A. 2004. Development of Sclerotinia stem rot in potato fields in south-central Washington. Plant Dis. 88:419-423.

Sclerotinia stem rot, caused by *Sclerotinia sclerotiorum*, is a recurrent disease on potato in the Columbia Basin of Washington. The role of ascospores in association with disease onset and stem rot incidence in commercial fields and the role of flower blossoms on plant infection were investigated in 10 fields over 2 years. Ascospores of *S. sclerotiorum* were detected on a semiselective medium over several weeks, with a peak in number of ascospores near initial full bloom. A high proportion of blossoms at initial full bloom were contaminated with *S. sclerotiorum* prior to blossom fall in most fields. Stem lesions occurred after row closure and blossom drop, and were associated with mycelial mats growing from contaminated blossoms that had dropped on plant stems in the plant canopy or blossoms that dropped on the ground and stems contacting the ground. Incidence of Sclerotinia stem rot was reduced significantly when blossoms were removed from plants before blossom drop. Flower blossoms were shown to be a paramount bridge between airborne ascospores of *S. sclerotiorum* and stem infection in the potato canopy.

Sclerotinia sclerotiorum (Lib.) de Bary is a cosmopolitan, homothallic and necrotrophic, ascomycetous fungus dispersed by airborne ascospores and soilborne sclerotia (17). Commercial crops susceptible to *S. sclerotiorum* and grown in the Columbia Basin of south-central Washington include potato, bean, pea, carrot, mint, cucurbits, and *Brassica* spp. (16). Reports on the economic impact of *S. sclerotiorum* on potato are scarce. Although some reports note severe damage (14), researchers in the U.S. Pacific Northwest (PNW) were not able to relate significant potato yield losses with disease incidence (11).

In the Columbia Basin, potato plants are grown in close proximity to other *S. sclerotiorum* susceptible crops or on fields where susceptible crops were grown the previous year. All potato cultivars (determinate and indeterminate growth habits) grown are susceptible to *S. sclerotiorum*

(unpublished data) and are mostly grown under center-pivot sprinkler irrigation at a density of 35,000 to 45,000 plants/ha. High crop density, close row width, and excess nitrogen fertilization were linked to increased stem rot incidence in several crops (4,13).

One to three fungicide applications usually are made to manage *S. sclerotiorum* on potato in the Columbia Basin using four registered fungicides: iprodione, dichloran, quintozene (synonym: pentachloronitrobenzene), and fluazinam. Fungicide label recommendations advise starting applications prior to row closure (i.e., complete covering of furrow between rows) to achieve the greatest stem coverage, with control often deemed inadequate by growers.

Ascospores of *S. sclerotiorum* are a major source of inoculum in bean, canola, and soybean where aerial parts are affected (2,3,18); whereas, in sunflower, the direct mycelial germination of sclerotia affects roots and crowns (6). In potato, intrafield apothecia are thought to supply most of the ascospore inoculum (15), which production coincided with the senescence of lower leaves following row closure (14). However, quantitative data are not available in support of ascospores being the major source of infection in potato.

Ascospores are unable to infect living tissue, but they saprophytically colonize flowers and senescing tissues that then are used as energy sources to infect green tissues (1,2,8,15,18). Inoculation of healthy and senescing potato leaves using ascospores released from apothecia inverted over the green tissue in moist chambers was not found to cause infection of potato tissue (unpublished data). Remov-

ing flowers from the crop canopy prevented stem rot occurrence in bean "even under ideal conditions" for infection (1).

The objectives of this study were to (i) evaluate the role of ascospores by capture on a semiselective medium on top of the plant canopy in association with disease onset and stem rot incidence in commercial fields, and (ii) identify the role of potato blossoms in the disease cycle of *S. sclerotiorum* on potato.

MATERIALS AND METHODS

Study area. Six commercial potato fields were selected for study in 2001 (fields 1 to 6) and four fields were selected in 2002 (fields 7 to 10) (Table 1). The indeterminate cvs. Russet Burbank, Gem Russet, Ranger Russet, Alturas, and Umattilla Russet and the determinate cv. Shepody were used. Cultivars did not differ for lesion size and latent period when inoculated with *S. sclerotiorum* in a preliminary study in the greenhouse (unpublished data). Fields were at least 53 ha in size, planted with certified seed tubers from mid-March through April of the respective years (Table 1), and were irrigated by center-pivot systems. Field 5 was planted late in early May, making it a late-planted field (Table 1). All fields were not planted with potato for at least three growing seasons preceding the year of study. Rotational crops planted in these fields were corn, onion, and wheat. Potato generally is not planted after potato; therefore, none of the studied fields could be used for both years of the study. Soils of all studied fields were sandy to sandy-loam deep soils and fields had been treated with Vapam (metam sodium, 378.5 liters/ha; AMVAC Chemical Corp., Newport Beach, CA) or Telone C17 (1,3-dichloropropene + chloropicrin, 130 liters/ha; Dow AgroSciences, Indianapolis, IN) prior to potato planting. Neither fumigant is known to reduce the incidence of Sclerotinia stem rot on potato in the Columbia Basin.

All fields were treated with fungicides by growers prior to row closure, following label recommendations, except for fields 9 and 10, which were treated at full bloom of primary flower clusters (initial full bloom will be used in the rest of the article) with fluazinam (Omega 500F, 0.5 liters/ha; Syngenta Crop Protection, Greensboro, NC), following the request of the authors. Fields 1, 2, 3, 4, 5, 6, 7, and 8 were treated with a mixture of iprodione and quintozene (Rovral 4F, 1.7 liters/ha; Bayer Crop-

Corresponding author: Z. K. Atallah
E-mail: atila@plantpath.wisc.edu

Present address of Z. K. Atallah: Department of Plant Pathology, University of Wisconsin, Madison 53706.

Financial and material support provided by the Washington State Potato Commission.

Plant Pathology New Series 0361, Department of Plant Pathology, College of Agriculture and Home Economics Research Center, Project No. 0678, Washington State University, Pullman 99164-6430.

Accepted for publication 7 December 2003.

Publication no. D-2004-0210-02R
© 2004 The American Phytopathological Society

Science, Research Triangle Park, NC; and Blocker 4F, 2.4 liters/ha, AMVAC). Fungicides were applied through the irrigation systems in all fields, with approximately 0.9 cm water/ha. One wedge of approximately 10 ha was left untreated with fungicides in fields 1 and 7, but each wedge otherwise was treated similarly to the rest of the field.

Detection and quantification of inoculum. Petri dishes containing a semiselective medium ("blue plates") were placed "face up" on top of plant canopies to capture airborne ascospores (5,7,19). Petri dishes were exposed for approximately 30 min between 9:00 a.m. and 2:00 p.m., coinciding with the diurnal peak in ascospore release as recorded in the literature (5,7,10). In all, 8 to 12 petri dishes were positioned at 40-m intervals along a randomly selected transect (sample transect) over the length of the field radius, including the last row at the edge of the field and the center of the circle. The number of petri dishes exposed depended on the radius of individual fields. Petri dishes were exposed weekly starting 3 weeks following plant emergence. Exposed petri dishes were incubated at 25°C for 3 to 4 days in the dark. Growing colonies were examined daily to identify *S. sclerotiorum*. Colonies of *S. sclerotiorum* usually were recognized within 3 to 4 days and were characterized by the absence of conidia, presence of whitish mycelium, and a color shift to yellow of the culture (19). Colonies from ascospores in petri dishes were counted in field 3, 7, 8, 9, and 10. Presence or absence of colonies from ascospores was recorded for fields 1, 2, 4, 5, and 6.

The number of ascospores captured was obtained by counting the number of *S.*

sclerotiorum colonies obtained on the semiselective medium, assuming that all viable ascospores captured germinated and that each colony resulted from a single ascospore. One-way analysis of variance (ANOVA) using PROC GLM in SAS (version 8.1; SAS Institute, Cary, NC) was used to analyze the number of captured ascospores (observational unit) among collection locations along sample transects within a field on a given date and among collection dates for all fields. The latter two factors were analyzed separately.

At initial full bloom, five flower clusters were collected from all fields in addition to the exposure of petri dishes, in the same locations where petri dishes were exposed. Initial full bloom was defined as the bloom of >90% flower blossoms on primary cymes on plants throughout the field; inflorescences develop on top of potato canopies. Initial full bloom generally occurred 7 to 10 days following row closure depending on cultivar and growing conditions. Two flowers per inflorescence (total, 10 blossoms/field) were plated on the semiselective medium to test for the presence of *S. sclerotiorum*, without surface disinfections.

Carpogenic germination was monitored in 80 m of row along the sample transect and near the pivot center in every field. The soil surface around the crowns of potato plants was scrutinized weekly for the presence of apothecia.

One-way ANOVA using PROC GLM was used to analyze the number of ascospores captured on the semiselective medium (observational unit) among collection locations and dates within a field.

Quantification of disease incidence. Potato plants along the sample transect

were observed weekly for disease onset starting at plant emergence. At the first appearance of stem rot symptoms, stems with lesions were counted in two rows (perpendicular to the sample transect) of 12 plants per row at the same sites where petri dishes were exposed. Numbers of infected stems and not lesions per individual stem were recorded because lesions coalesce, potentially causing record-keeping difficulties. Two weeks after the first assessment, a second disease assessment was performed on a second set of two rows of 12 plants adjacent to the previously assessed rows. Different but contiguous rows were used because opening rows disrupted the canopy microclimate during the first disease assessment. Only two disease assessments were carried out in each field because of harvest or increase in bacterial rot incidence, which hampered proper identification of rot agent. Sample transects in fields 1 and 7 were located in the untreated control wedges of both fields but disease assessments were carried out in both wedges.

Blossom removal. To help determine the role of blossoms in disease development, blossoms were removed from plants or fungicides were applied to plant canopies in three rows by 10-m-long plots in field 7. The tests were conducted in the wedge-shaped section of the field not treated with fungicides. Flower clusters carrying flower primordia and flowers were handpicked as they became apparent over a 3-week period. Fungicides iprodione (Rovral 4F, 2.4 liters/ha; Bayer CropScience) and fluazinam (Omega 500F, 0.5 liters/ha; Syngenta Crop Protection) were applied to the canopy of plots at initial full bloom in field 7. The experiment

Table 1. Location, crop, and Sclerotinia stem rot information from commercial potato fields in the Columbia Basin of Washington, investigated in 2001 and 2002

Field, locations ^a	Cultivar ^v	Date ^s				Stems infected ^t		Fung. ^w	Asco. ^x	Inf. (%) ^y	Apoth. ^z
		Planting	Closure	Bloom	Onset	Onset	2 weeks				
1, 50 km Southwest Pasco	Gem	3 Apr 01	10 Jun 01	18 Jun 01	22 Jun 01	170	399	IQ	Yes	100	Yes
2, 50 km Southwest Pasco	Ranger	5 Apr 01	10 Jun 01	18 Jun 01	...	0	0	IQ	Yes	100	Yes
3, 10 km North Pasco	Shepody	23 Mar 01	13 Jun 01	18 Jun 01	28 Jun 01	330	287	IQ	Yes	100	Yes
4, 10 km North Pasco	Ranger	2 Apr 01	19 Jun 01	25 Jun 01	29 Jun 01	220	203	IQ	Yes	100	Yes
5, 10 km West Warden	Gem	5 May 01	10 Jul 01	19 Jul 01	25 Jul 01	90	64	IQ	Yes	100	Yes
6, 5 km North Eltopia	Alturas	16 Apr 01	25 Jun 01	2 Jul 01	...	0	0	IQ	Yes	100	No
7, 10 km North Pasco	Ranger	1 Apr 02	12 Jun 02	17 Jun 02	27 Jun 02	211	344	IQ	Yes	100	Yes
8, 10 km North Pasco	Ranger	1 Apr 02	12 Jun 02	17 Jun 02	27 Jun 02	57	297	IQ	Yes	100	Yes
9, 15 km West Basin City	Burbank	12 Apr 02	18 Jun 02	25 Jun 02	...	0	0	FI	No	0	No
10, 15 km West Basin City	Umatilla	13 Apr 02	18 Jun 02	28 Jun 02	...	0	0	FI	Yes	0	No

^s Planting = planting date, Closure = row closure date, Bloom = date of initial full bloom, and Onset = date of disease onset. Disease onset in field 5 is based on aerial symptoms appearance. Lesions induced by direct mycelial germination from sclerotia were observed on 5% of plants prior to row closure.

^t Number of stems infected with *S. sclerotiorum* per 120 plants at disease onset and 2 weeks following onset.

^u Fields 1 and 2 were adjacent to each other, but sample transects were separated by 1 km. Fields 3, 4, 7, and 8 were located on the same farm; sample transects in fields 3 and 4 were separated by 0.5 km and sample transects in fields 7 and 8 were separated by 1 km.

^v Cvs. Russet Burbank (Burbank), Gem Russet (Gem), Ranger Russet (Ranger), Alturas, and Umatilla Russet (Umatilla) are indeterminate and cv. Shepody is determinate.

^w Fung. = fungicides applied prior to row closure in fields 1 through 8, and at initial full bloom in fields 9 and 10, through center-pivot irrigation systems; IQ = iprodione + quintozene and FI = fluazinam.

^x Asco. = ascospores captured: presence or absence of ascospores in petri dishes of semiselective medium placed on top of plant canopy.

^y Inf. = blossom infection: detection of *S. sclerotiorum* ascospores on potato blossoms by plating on semiselective medium.

^z Apoth. = apothecia observed: occurrence of *S. sclerotiorum* apothecia in 80 m of row.

was repeated in 2003 in a Ranger Russet field, 10 km north of Pasco, WA (field 11), where only fluazinam (Omega 500F, 0.5 liters/ha; Syngenta Crop Protection) at initial full-bloom was compared with flower removal. Fungicides were applied using a CO₂ pressurized sprayer at a rate of 281 liters/ha at 210 kPa, using a ConeJet hollow cone nozzle TXVS-18 (R&D Sprayers Inc., Opelousas, LA). Blossoms were not removed and the canopy was not sprayed with fungicide in the nontreated control plots. The experiments were arranged in a randomized complete block design where treatments of fungicide applications or blossom removal were replicated four times. Disease incidence was assessed on 30 plants in the middle row by counting the number of stems with lesions 3 weeks after fungicide application. Data for incidence of infected stems were analyzed by one-way ANOVA using PROC GLM in SAS. Fungicides and flower removal served as treatments and lesion numbers were considered the observational unit. Fisher's protected least significant difference was used to compare treatment means.

RESULTS

Detection and quantification of inoculum. Ascospores were captured in 9 of the 10 scrutinized fields. Numbers of captured ascospores differed significantly among collection dates in all fields ($P < 0.05$). The greatest ascospore numbers were captured beginning about a week before and continuing a few days after initial full bloom (Fig. 1). Ascospore numbers did not differ significantly among trapping sites along the sample transect within all fields ($P > 0.05$). Ascospores that produced colonies of *S. sclerotiorum* were not captured on the semiselective medium or blossoms during the entire growing season in field 9.

Of the flowers collected at initial full bloom from all fields except fields 9 and 10, 100% were contaminated (i.e., ascospores were present on the flower surface but had not yet caused infection and subsequent colonization of blossom tissues, which typically occurs following blossom senescence) with *S. sclerotiorum*, as verified by mycelial growth from the blossoms plated on the semiselective medium. Contaminated blossoms were not detected in fields 9 and 10.

Carpogenic germination coincided with row closure in fields 1, 2, 3, 4, 5, 7, and 8, where 7 to 11 apothecia were counted in 80 m of row along the sample transect. No apothecia were observed in fields 6, 9, and 10.

Quantification of disease incidence. In fields 1, 3, 4, 5, 7, and 8, stem rot initially appeared on potato stems 14 to 20 days after row closure and 5 to 7 days following blossom fall. This coincided with stems touching the ground. Mycelial mats of *S. sclerotiorum* were observed to colonize

senesced blossoms on the ground and on stems in the plant canopy following initial full bloom and blossom fall. Lesions developed on stems contacted by colonized blossoms. Flowers showing a white mycelial growth were collected from the soil surface and plated on the semiselective medium and, in every instance, the blossoms were found to be colonized by *S. sclerotiorum*.

Healthy stems became infected when contacting infected tissues, but rarely did whole plants die, unless infections occurred in the crown area at the base of plants. In field 5, stem rot lesions were detected 14 days before row closure and involved 5% of the plants. Excavating soil from around wilted plants revealed lesions in the crown area about 1 to 2 cm below soil level. Hyphae of *S. sclerotiorum* were observed emerging from sclerotia germinating within 2 cm of the lesion. Infected stems eventually were girdled and killed as a result of such infections.

Disease progress curves increased rapidly after symptom appearance in fields 1, 3, 4, 5, 7, and 8. In fields 1, 7, and 8, disease incidence increased significantly ($P < 0.05$) between the first and second disease assessments (Fig. 2). Conversely, in fields 3, 4, and 5, disease incidence did not vary significantly between the first and second disease assessments ($P > 0.05$; Fig. 2).

Fungicide applications prior to row closure did not prevent the occurrence of stem rot in fields 1, 3, 4, 5, 7, and 8 (Fig. 2). Stem rot incidence was not different between treated and control areas in fields 1 and 7; applications of fungicides at initial full bloom in fields 7 and 11 resulted in a significant reduction in disease incidence in treated plots compared with nontreated controls (Table 2).

Stem rot did not develop in fields 2, 6, 9, and 10. The soil surface in fields 2 and 6 was littered with colonized blossoms but stems did not become infected. *S. sclerotiorum* was not detected on blossoms attached to plants and on the soil surface in field 9, where ascospores were not detected, and field 10, where ascospores repeatedly were captured on semiselective medium but no evidence of flower contamination was observed.

Blossom removal. Incidence of Sclerotinia stem rot (expressed as number of lesions per 30 plants) in plots where flowers were removed in fields 7 and 11 was significantly less than in the untreated control plots ($P < 0.05$), but was not significantly different from disease incidence in plots treated with fungicides at initial full bloom ($P > 0.05$; Table 2).

DISCUSSION

Epidemics of Sclerotinia stem rot of potato in six studied fields in the Columbia Basin primarily were initiated by ascospores produced from May to mid-July, and blossoms were crucial for the develop-

ment of Sclerotinia stem rot on potato. The role of potato blossoms and the timing of blossom contamination, whether on the plant canopy or fallen on the ground, were not previously known. Blossoms acted as ascospore receptacles and became contaminated while still attached to the plant canopy. Removing blossoms from the plant canopy gave further evidence for the role of blossoms in the infection of potato stems. This is similar to studies on canola, where ascospore presence on flower petals was positively correlated with disease occurrence (9,20), and on bean, where blossoms were described as "essential intermediaries" between the fungus and the host (2). Ascospores evidently remained inactive until blossoms fell and senesced, after which they rapidly colo-

Fig. 1. Mean number of ascospores captured on top of potato plant canopies and using the *Sclerotinia sclerotiorum* semiselective medium in field 3 in 2001 and fields 7, 8, and 10 in 2002. RC = row closure, IB = initial full bloom, DO = disease onset.

nized blossom tissues (18,21) if within the humid microclimate of the crop canopy. Mycelium of *S. sclerotiorum* then was transferred from colonized blossoms to green stems as blossoms fell on foliage and when stems came in contact with colonized blossoms on the soil surface. Disease transmission occurred 14 to 20 days after row closure depending on cultivars and cultural practices.

Blossom colonization was observed on the soil surface in fields 2 and 6, following blossom drop, but disease failed to occur because plants did not lay on the ground until a month after initial full bloom. This lag period allowed for the disintegration of the mycelial mats on the soil surface, which disappeared 7 to 10 days following development. Cultural and fertilization practices that limit stem growth or encourage an erect plant architecture may be beneficial as disease escape strategies.

Small numbers of apothecia were observed and counted in seven fields; also, ascospores were captured prior to the appearance of apothecia in respective fields, and at the very edge of fields. These observations reveal a need for further research to ascertain the origin of inoculum for potato fields, especially in light of reports regarding *S. sclerotiorum* epidemics in bean and rapeseed initiated by ascospores released outside studied fields (1,2,21).

Removal of blossoms or fungicide applications at initial full bloom reduced the

number of *Sclerotinia* stem rot lesions (Table 2). Fungicide applications in the experimental wedges in fields 7 and 11 at initial full bloom reduced stem incidence because blossom contamination and subsequent colonization of blossoms by *S. sclerotiorum* ascospores was reduced at the time of peak ascospore release. Even though no control plots were established in field 10, the application of Fluazinam at initial full bloom likely prevented colonization of blossoms and subsequent stem rot development, especially because viable ascospores were captured on the semiselective medium and the fungus was not recovered from collected blossoms. Applications of fungicides prior to row closure did not reduce incidence of *Sclerotinia* stem rot, as illustrated by disease progress curves in the six fields where potato stem rot occurred (Table 1; Fig. 2). Blossoms had not developed and were not subsequently protected by fungicide applications. Similar observations were made when bean flowers were protected with benomyl at full bloom, which prevented white mold development even under optimal environmental conditions (2,18). Applications of benomyl made after full bloom failed to provide effective stem rot control because flowers had already acquired the inoculum (12,13).

The rapid reduction in the numbers of captured ascospores in early July indicates that planting dates and cultivar choices

may influence stem rot incidence. Fields planted early would be subject to extended ascospore-release periods, whereas those planted later likely escape the ascospore releases, which tend to decrease markedly in July. Additional research is needed to evaluate the effect of planting date on *Sclerotinia* stem rot incidence in potato.

The epidemiology of *Sclerotinia* stem rot in potato seems highly associated with crop management decisions as a result of timing and quantity of ascospore release periods and a limited direct mycelial germination. Even though experiments were not conducted to compare various cultivars with regard to their growth habit, it is likely that the density of canopy and cultivar growth habit play a crucial role in the ability to economically manage *Sclerotinia* stem rot in potato.

This study demonstrated the role of potato flower blossoms in the disease cycle of *Sclerotinia* stem rot on potato. Fallen and senesced flowers served as a crucial bridge between airborne ascospores of *S. sclerotiorum* and potato stems, while minimal infection was found to be induced by the direct mycelial germination of sclerotia. Application of fungicide at initial full bloom effectively reduced the incidence of *Sclerotinia* stem rot in replicated plots in 2002 and 2003. Timing fungicide applications to correspond with initial full bloom will likely be more effective in disease control than the current practice of applying fungicide at row closure. However, several application timings, from row closure through potato blossoming, need to be compared directly in field experiments to determine the most appropriate application timing.

ACKNOWLEDGMENTS

We thank T. F. Cummings for assistance; F. Dugan, G. Grove, and X. Chen for critical reviews; and the editor and anonymous reviewers of *Plant Disease* for their reviews, suggestions, and comments.

Table 2. Incidence of potato stems infected with *Sclerotinia sclerotiorum* when flower blossoms were removed from the plant canopy or treated with fungicide at initial full bloom^v

Treatment	2002 ^w	2003 ^x
Iprodione	27 b	...
Fluazinam	16 b	10 b
Blossom removal ^y	25 b	3 c
Control ^z	87 a	44 a
LSD ($P \leq 0.05$)	11.2	6.98

^v Means within a column ($n =$ five replicates) followed by the same letter are not significantly different using Fisher's protected least significant difference (LSD; $P \leq 0.05$).

^w Experiment conducted in field 7 in 2002, planted with cv. Ranger Russet.

^x Experiment conducted in field 11 in 2003, planted with cv. Ranger Russet.

^y Blossoms and blossom primordia removed from primary cyme over 3 weeks.

^z No fungicides applied to plant canopies.

Fig. 2. Disease progress curves of *Sclerotinia* stem rot in fields 1, 3, 4, and 5 in 2001 and fields 7 and 8 in 2002.

LITERATURE CITED

1. Abawi, G. S., and Grogan, R. G. 1974. Ascospores of *Whetzelinia sclerotiorum* as primary inoculum causing white mold of beans in New York. (Abstr.) *Phytopathology* 64:578.
2. Abawi, G. S., Polach, F. J., and Molin, W. T. 1975. Infection by ascospores of *Whetzelinia sclerotiorum*. *Phytopathology* 65:673-678.
3. Boland, G. J., and Hall, R. 1988. Epidemiology of *Sclerotinia* stem rot of soybean in Ontario. *Phytopathology* 78:1241-1245.
4. Grau, C. R., and Radke, V. L. 1984. Effects of cultivars and cultural practices on *Sclerotinia* stem rot of soybeans. *Plant Dis.* 68:56-58.
5. Gutierrez, W. A., and Shew, H. D. 1998. Identification and quantification of ascospores as the primary inoculum for collar rot of greenhouse-produced tobacco seedlings. *Plant Dis.* 82:485-490.
6. Huang, H. C., and Dueck, J. 1980. Wilt of sunflower from infection by mycelial-germinating sclerotia of *S. sclerotiorum*. *Can. J. Plant Pathol.* 2:47-52.
7. Hudyncia, J., Shew, H. D., Cody, B. R., and Cubeta, M. A. 2000. Evaluation of wounds as a factor to infection of cabbage by ascospores of *Sclerotinia sclerotiorum*. *Plant Dis.* 84:316-320.
8. Keay, M. A. 1939. A study of certain species of the genus *Sclerotinia*. *Ann. Appl. Biol.* 26:227-246.
9. Lefol, C., and Morrall, R. A. A. 1996. Immunofluorescent staining of *Sclerotinia* ascospores on canola petals. *Can. J. Plant Pathol.* 18:237-241.
10. McCartney, H. A., and Lacey, M. E. 1992. Release and dispersal of *Sclerotinia* ascospores in relation to infection. *Brighton Crop Prot. Conf. Pests Dis.* 3A-2:109-116.
11. Miller, T. D., and Miller, J. S. 2001. *Sclerotinia* white mold of potato. Pages 185-186 in: *Proc. 2001 Idaho Winter Commodity Schools*, Aberdeen, ID.
12. Morton, J. G., and Hall, R. 1989. Factors determining the efficacy of chemical control of white mold in white bean. *Can. J. Plant Pathol.* 11:297-302.
13. Natti, J. J. 1971. Epidemiology and control of bean white mold. *Phytopathology* 61:669-674.
14. Partyka, R. E., and Mai, W. F. 1961. Effects of environment and some chemicals on *Sclerotinia sclerotiorum* in laboratory and potato field. *Phytopathology* 52:766-770.
15. Powleson, M. L. 2001. White mold. Pages 47-48 in: *Compendium of Potato Diseases*. 2nd ed. W. R. Stevenson, R. Loria, G. D. Franc, and D. P. Weingartner, eds. American Phytopathological Society Press, St. Paul, MN.
16. Pscheidt, J. W., and O'camb, C. M. eds. 2002. *Pacific Northwest Plant Disease Management Handbook*. Oregon State University, Corvallis.
17. Purdy, L. H. 1979. *Sclerotinia sclerotiorum*: history, diseases and symptomatology, host range, geographic distribution, and impact. *Phytopathology* 69:875-880.
18. Steadman, J. R. 1983. White mold—a serious yield-limiting disease of bean. *Plant Dis.* 67:346-350.
19. Steadman, J. R., Marcinkowska, J., and Rutledge, S. 1994. A semi-selective medium for isolation of *Sclerotinia sclerotiorum*. *Can. J. Plant Pathol.* 16:68-70.
20. Turkington, T. K., and Morrall, R. A. A. 1993. Use of petal infestation to forecast *Sclerotinia* stem rot of canola: the influence of inoculum variation over the flowering period and canopy density. *Phytopathology* 83:682-689.
21. Williams, J. R., and Stelfox, D. 1979. Dispersal of ascospores of *Sclerotinia sclerotiorum* in relation to *Sclerotinia* stem rot of rapeseed. *Plant Dis. Rep.* 63:395-399.